Autoimmune Pheromonal and Hormonal Modification Verifiable by Effect Upon Insect Behavior – Implications for Autoimmune Disorders

19 July 2025
Simon Edwards
Research Acceleration Initiative

Introduction

Allow a mosquito to bite a human on the arm and wait a few days for the itching and irritation to settle down. Give another mosquito the opportunity to bite the same arm and a complete different mosquito will have an enhanced probability of selecting the same area bitten by the first mosquito. The first mosquito's choice of bite location was likely pure happenstance, but when the second mosquito chooses the same location, this is not a coincidence. This is the result of the induction of the human immune system into creating altered versions of its own pheromones.

Abstract

When an insect such as a mosquito bites a human, toxins are introduce which produce an autoimmune response. This response leads to itching, redness and swelling. Although the way in which the human immune system attacks tissues and organ systems has been extensively studied, the way in which the immune system may attack free-floating hormones within interstitial fluids and in the blood stream has been largely neglected.

For example, many patients with Hashimoto's Thyroiditis present with a false negative on standard tests for the condition because, I would posit, it is the triiodothyronine which is being attacked or modified after production in addition to the crippling of the ability of the thyroid to produce the T4 precursor. A condition called Reverse T3 Syndrome has been identified which is defined as the reversal of the chirality of triiodothyronine, which renders it inert. This condition, however, is rare, as it requires a mutation in the mode of function of the conversion mechanism which converts T4 into T3. It stands to reason that there may be other, undiscovered syndromes which have as their root cause more subtle modifications of triiodothyronine, post-production. A great many patents who do not respond fully to T4 replacement have been helped by augmentation of both T4 and T3, but even this approach provides only temporary relief as the fundamental problem is that the immune system is appending a molecule to T3 which alters its metabolic function. Much more likely in those patents who report incomplete symptom resolution with T4 supplements is some intermediary effect upon triiodothyronine which appends an inert molecule to the T3 which reduces the sensitivity of tissues to the hormone. Before long, porcine triiodothyronine becomes affected by this autoimmune corruption of the molecule despite slight differences in structure.

This likelihood of this hypothesis concerning Hashimoto's Thyroiditis can be supported by studying the behavior of mosquitoes and their choice of bite location. Natural human pheromones can be detected by insects (in addition to insectoid pheromones) and human pheromones modified by an immune

response to the presence of toxins introduced by a mosquito may play a role not yet identified by researchers in attracting mosquitoes.

Many patients with autoimmune conditions such as Lupus or Sjorgren's Syndrome report a greater than average number of mosquito and tick bites, perhaps because the same mechanism which mosquitoes have developed over the eons to demarcate known quality food sources for later utilization is constantly at play throughout their bodies. Insects likely perceive individuals with certain autoimmune disorders as having been previously bitten and therefore treat them as ideal food sources.

Conclusion

If the preceding is true and the human immune system can modify its own pheromones and hormones as a result of inappropriate autoimmune activity, it stands to reason that many medical conditions may have as their root cause an autoimmune response which alters hormones and other chemicals when they are already in transit in the blood stream.